<u>In the Claims:</u>

Cancel Claims 1-36 and add new claims 37-82 as shown below.

37. A method for inhibiting the formation of β-amyloid plaques in the brain of a human, the method comprising:
    a) providing a β-amyloid epitope; and
    b) administering the epitope of step a) to the human under conditions appropriate for the stimulation of an immune response directed toward the epitope, the immune response being characterized by the generation of circulating antibodies which bind specifically to the epitope present on endogenous β-amyloid in the human.

38. The method of Claim 37 wherein the epitope of step a) is administered in an adjuvant formulation.

39. The method of Claim 38 wherein the adjuvant formulation comprises an alum adsorption.

40. The method of Claim 38 wherein the adjuvant formulation comprises oil emulsion.

41. The method of Claim 37 wherein the binding of circulating antibodies to endogenous β-amyloid detectably alters the equilibrium distribution of free β-amyloid in circulation versus free β-amyloid in the brain of the human.

42. The method of Claim 37 wherein the epitope of β-amyloid is linked to an immunogenic carrier moiety.

43. The method of Claim 42 wherein the immunogenic carrier moiety is diphtheria toxoid.

44. The method of Claim 42 wherein the immunogenic carrier moiety is hepatitis B core antigen.

45. The method of Claim 37 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-43}$.

46. The method of Claim 37 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-42}$.

47. The method of Claim 37 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-41}$.

48. The method of Claim 37 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-40}$.

49. The method of Claim 37 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the N-terminal region of the β-amyloid peptide $A\beta_{1-43}$.

50. The method of Claim 37 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the central region of the β-amyloid peptide $A\beta_{1-43}$.

51. The method of Claim 37 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the C-terminal region of β-amyloid peptide.

52. The method of Claim 37 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to immobilized β-amyloid peptides in an *in vitro* binding assay, the immobilized β-

amyloid peptides being selected from the group consisting of: $A\beta_{1-16}$, $A\beta_{14-25}$, $A\beta_{34-43}$, $A\beta_{1-40}$ and $A\beta_{1-43}$.

53. The method of Claim 37 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to β-amyloid in solution.

54. A method for inhibiting the formation of β-amyloid plaques in the brain of a human, the method comprising:
   a) providing a plurality of peptide fragments derived from β-amyloid peptide $A\beta_{1-43}$, each peptide fragment comprising one or more β-amyloid epitopes; and
   b) administering the plurality of peptide fragments of step a) to the human under conditions appropriate for the stimulation of an immune response directed toward the β-amyloid epitopes, the immune response being characterized by the generation of circulating antibodies which bind specifically to one or more epitopes present on endogenous β-amyloid in the human.

55. The method of Claim 54 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to immobilized β-amyloid peptides in an *in vitro* binding assay, the immobilized β-amyloid peptides being selected from the group consisting of: $A\beta_{1-16}$, $A\beta_{14-25}$, $A\beta_{34-43}$, $A\beta_{1-40}$ and $A\beta_{1-43}$.

56. The method of Claim 54 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to β-amyloid in solution.

57. The method of Claim 54 wherein at least one epitope is provided as a peptide fragment of β-amyloid, the peptide

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fragment being derived from the N-terminal region of the β-amyloid peptide $A\beta_{1-43}$.

58. The method of Claim 54 wherein at least one epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the central region of the β-amyloid peptide $A\beta_{1-43}$.

59. The method of Claim 54 wherein at least one epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the C-terminal region of β-amyloid peptide.

60. A method for inhibiting the formation of β-amyloid plaques in the brain of a human, the method comprising:
   a) providing an antibody which binds specifically to an epitope of β-amyloid peptide; and
   b) delivering the antibody of step a) into the circulation of the human at concentrations sufficient to detectably alter the equilibrium distribution of free β-amyloid peptide in circulation versus free β-amyloid peptide in the brain of the human.

61. The method of Claim 60 wherein the antibody has the ability to inhibit the formation of β-amyloid plaques.

62. The method Claim 60 wherein the antibody has the ability to disaggregate preformed β-amyloid plaques.

63. The method of Claim 60 wherein the antibody has the ability to hydrolytically cleave β-amyloid.

64. A vaccine composition comprising a β-amyloid epitope in an adjuvant formulation.

65. The vaccine composition of Claim 64 wherein the β-amyloid epitope is linked to an immunogenic carrier moiety.

66. The vaccine composition of Claim 65 wherein the immunogenic carrier moiety is diphtheria toxoid.

67. The vaccine composition of Claim 65 wherein the immunogenic carrier moiety is hepatitis B core antigen.

68. The vaccine composition of Claim 64 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-43}$.

69. The vaccine composition of Claim 64 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-42}$.

70. The vaccine composition of Claim 64 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-41}$.

71. The vaccine composition of Claim 64 wherein the epitope is provided as β-amyloid peptide $A\beta_{1-40}$.

72. The vaccine composition of Claim 64 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the N-terminal region of the β-amyloid peptide $A\beta_{1-43}$.

73. The vaccine composition of Claim 64 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide fragment being derived from the central region of the β-amyloid peptide $A\beta_{1-43}$.

74. The vaccine composition of Claim 64 wherein the epitope is provided as a peptide fragment of β-amyloid, the peptide

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fragment being derived from the C-terminal region of the β-amyloid peptide $A\beta_{1-43}$.

75. The vaccine composition of Claim 64 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to immobilized β-amyloid peptides in an *in vitro* binding assay, the immobilized β-amyloid peptides being selected from the group consisting of: $A\beta_{1-16}$, $A\beta_{14-25}$, $A\beta_{34-43}$, $A\beta_{1-40}$ and $A\beta_{1-43}$.

76. The vaccine composition of Claim 64 which results in the stimulation of an immune response which includes the production of antibodies which bind specifically to β-amyloid.

77. The vaccine composition of Claim 64 wherein the β-amyloid is free in solution.

78. The vaccine composition of Claim 64 wherein the β-amyloid is aggregated.

79. A method for inhibiting the formation of β-amyloid plaques in the brain of a human, the method comprising:
   a) providing an antibody which binds specifically to an epitope of β-amyloid peptide; and
   b) delivering the antibody of step a) by direct infusion into the brain of the human.

80. The method of Claim 79 wherein the antibody has the ability to inhibit the formation of β-amyloid plaques.

81. The method of Claim 79 wherein the antibody has the ability to disaggregate preformed β-amyloid plaques.

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